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NEWS 1		Web Page for STN Seminar Schedule - N. America
NEWS 2	JUL 02	LMEDLINE coverage updated
NEWS 3	JUL 02	SCISEARCH enhanced with complete author names
NEWS 4	JUL 02	CHEMCATS accession numbers revised
NEWS 5	JUL 02	CA/CAplus enhanced with utility model patents from China
NEWS 6	JUL 16	CAplus enhanced with French and German abstracts
NEWS 7	JUL 18	CA/CAplus patent coverage enhanced
NEWS 8	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 9	JUL 30	USGENE now available on STN
NEWS 10	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS 11	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 12	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 13	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 14	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS 15	AUG 27	USPATOLD now available on STN
NEWS 16	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS 17	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS 18	SEP 13	FORIS renamed to SOFIS
NEWS 19	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 20	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 21	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 22	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 23	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 24	OCT 19	BEILSTEIN updated with new compounds
NEWS 25	NOV 15	Derwent Indian patent publication number format enhanced
NEWS 26	NOV 19	WPIX enhanced with XML display format

NEWS EXPRESS 19 SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.

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NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

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* * * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:18:34 ON 19 NOV 2007

=> fil reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 11:18:43 ON 19 NOV 2007

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 18 NOV 2007 HIGHEST RN 954747-20-7
DICTIONARY FILE UPDATES: 18 NOV 2007 HIGHEST RN 954747-20-7

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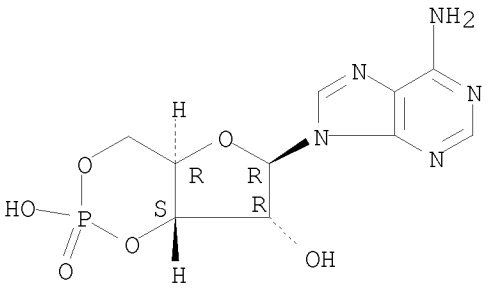
=> s 60-92-4/rn
L1 1 60-92-4/RN

=> d l1

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 60-92-4 REGISTRY
ED Entered STN: 16 Nov 1984
CN Adenosine, cyclic 3',5'-(hydrogen phosphate) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 4H-Furo[3,2-d]-1,3,2-dioxaphosphorin, adenosine deriv.
CN Adenosine 3',5'-cyclic phosphate (6CI)
OTHER NAMES:
CN 1: PN: US20040005997 TABLE: 1 claimed sequence
CN 3',5'-AMP
CN 45: PN: US20030109453 SEQID: 44 claimed sequence
CN Adenosine 3',5'-cyclophosphate

CN Adenosine 3',5'-monophosphate
CN Adenosine 3',5'-phosphate
CN Adenosine cyclic 3',5'-monophosphate
CN Adenosine cyclic monophosphate
CN cAMP
CN Cyclic 3',5'-adenylic acid
CN Cyclic 3',5'-AMP
CN Cyclic adenosine 3',5'-monophosphate
CN Cyclic adenosine 3',5'-phosphate
CN Cyclic AMP
CN NSC 143670
CN NSC 94017
FS STEREOSEARCH
DR 11002-78-1
MF C10 H12 N5 O6 P
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, PIRA, PROMT, PS, RTECS*, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL, USPATOLD, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

61882 REFERENCES IN FILE CA (1907 TO DATE)
352 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
61938 REFERENCES IN FILE CAPLUS (1907 TO DATE)
108 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.40	2.61

FILE 'CAPLUS' ENTERED AT 11:19:09 ON 19 NOV 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 19 Nov 2007 VOL 147 ISS 22
FILE LAST UPDATED: 18 Nov 2007 (20071118/ED)

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http://www.cas.org/infopolicy.html

```
=> s l1
L2       61938 L1

=> inhibitor and l2
INHIBITOR IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s inhibitor and l2
        557198 INHIBITOR
        560136 INHIBITORS
        874243 INHIBITOR
                 (INHIBITOR OR INHIBITORS)
L3       12950 INHIBITOR AND L2

=> s (vitamin C OR "L-Ascorbic acid")
        203963 VITAMIN
         58924 VITAMINS
        226993 VITAMIN
                 (VITAMIN OR VITAMINS)
       3722555 C
         43866 VITAMIN C
                 (VITAMIN(W)C)
       1612330 "L"
         87171 "ASCORBIC"
       4477714 "ACID"
       1601200 "ACIDS"
       4982630 "ACID"
                 ("ACID" OR "ACIDS")
         14724 "L-ASCORBIC ACID"
                 ("L"(W)"ASCORBIC"(W)"ACID")
L4       56140 (VITAMIN C OR "L-ASCORBIC ACID")

=> s l3 and l4
L5          18 L3 AND L4

=> s l5 and (ay<2002 or py<2002 or pry<2002)
       4191486 AY<2002
      21918241 PY<2002
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       3668602 PRY<2002
L6          14 L5 AND (AY<2002 OR PY<2002 OR PRY<2002)

=> d l6 1-5 ibib abs kwic

L6   ANSWER 1 OF 14  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2006:1228580  CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER:         145:500166
TITLE:                   Capillary membrane stabilization and reduction of
                         tissue injury through use of biodegradable
                         polysaccharides with antioxidants and/or other
                         chemicals
INVENTOR(S):             Zikria, Bashir A.; Zikria, Jemal Dean
PATENT ASSIGNEE(S):      USA
SOURCE:                  U.S. Pat. Appl. Publ., 12pp., Cont.-in-part of U.S.
                         Ser. No. 837,840.
                         CODEN: USXXCO
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     ---------------     ----   --------    --------------------    --------
     US 2006264357        A1    20061123    US 2005-213303          20050829 <--
     US 7041655           B1    20060509    US 1997-837840          19970422 <--
PRIORITY APPLN. INFO.:                      US 1997-837840      A2  19970422 <--
                                            US 1996-15963P      P   19960424 <--
AB   The invention provides a method of treating a human subject to prevent
     leakage of serum proteins from capillary endothelial junctions during a
     period of increased capillary permeability and at the same time preventing
     the harmful effects of free radicals on capillaries and surrounding
     tissues.  The method comprises administering to a subject an effective
     amount of a composition comprising at least one polysaccharide selected from
     hydroxyethyl starch, glycogen and dextran of varying mol. sizes, and at
     least one active agent selected from dehydroascorbic acid, von Willebrand
     Factor, Hb, polysaccharide-conjugated Hb, Cerovive, edaravone,
     dimethylthiourea, citicoline, poly(ADP-ribose) polymerase
     inhibitor, oxidant detoxification catalyst, adenosine 2a (A2a)
     receptor agonist, adenosine 1 (A1) receptor agonist, adenosine, inosine,
     xanthin oxidase inhibitor, polyethylene-glycol-modified albumin,
     ATP, histamine, taurine, simvastatin, atrial natriuretic peptide,
     sphinogosine 1-phosphate, apyrase, secretory leukocyte protease
     inhibitor, antithrombin III, adrenomedullin, i.v.
     immunologlobulin, sodium beta-aescin, Δ2-1,2,3-triazoline and
     aminoalkylpyridine, aromatase inhibitors, and neuropilin-1,
     polynitroxyl albumin, α-phenyl-N-tert-Bu nitrone and the antioxidant
     subgroup consisting of tocopherols, tocotrienols, carotenoids, minerals
     and mineral-containing organic compds., polyphenols, lipoic acids, transition
     metal ion-binding proteins, melatonin, hormones, polyamines, tamoxifen and
     its metabolites, and propofol.  The composition may further contain at least
     one member of the group of superoxide dismutase, glutathione peroxidase,
     catalase, hydroxyethyl rutoside, cyclic adenosine monophosphate and
     vitamin C.  The compns. contain the macromols. in a mol.
     size and concentration adequate to effectively stabilize the capillary
membrane.
     The stabilization effect is accompanied by a biophys. and biochem. process
     due to the adhesiveness and configuration of the macromols., and because
     of their size.  The treatment is benign as the macromols. and active
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agents are non-toxic and biodegradable.

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2006264357	A1	20061123	US 2005-213303	20050829 <--
	US 7041655	B1	20060509	US 1997-837840	19970422 <--
PRAI	US 1997-837840	A2	19970422 <--		
	US 1996-15963P	P	19960424 <--		

AB . . . least one active agent selected from dehydroascorbic acid, von Willebrand Factor, Hb, polysaccharide-conjugated Hb, Cerovive, edaravone, dimethylthiourea, citicoline, poly(ADP-ribose) polymerase inhibitor, oxidant detoxification catalyst, adenosine 2a (A2a) receptor agonist, adenosine 1 (A1) receptor agonist, adenosine, inosine, xanthin oxidase inhibitor, polyethylene-glycol-modified albumin, ATP, histamine, taurine, simvastatin, atrial natriuretic peptide, sphinogosine 1-phosphate, apyrase, secretory leukocyte protease inhibitor, antithrombin III, adrenomedullin, i.v. immunologlobulin, sodium beta-aescin, Δ2-1,2,3-triazoline and aminoalkylpyridine, aromatase inhibitors, and neuropilin-1, polynitroxyl albumin, α-phenyl-N-tert-Bu nitrone and the antioxidant subgroup consisting of tocopherols, tocotrienols, carotenoids, minerals and mineral-containing organic compds.,. . . contain at least one member of the group of superoxide dismutase, glutathione peroxidase, catalase, hydroxyethyl rutoside, cyclic adenosine monophosphate and vitamin C. The compns. contain the macromols. in a mol. size and concentration adequate to effectively stabilize the capillary membrane. The stabilization. . .

IT 50-81-7, Vitamin C, biological studies 51-45-6, Histamine, biological studies 51-48-9, Thyroxine, biological studies 53-00-9, 7α-Hydroxy-dehydroepiandrosterone 53-43-0, Dehydroepiandrosterone 56-65-5, Adenosine triphosphate, biological studies 58-61-7, Adenosine, biological studies 58-63-9, Inosine 59-02-9, α-Tocopherol 60-92-4 69-72-7, Salicylic acid, biological studies 70-51-9, Deferoxamine 71-44-3, Spermine 73-31-4, Melatonin 89-25-8, Edaravone 107-35-7, Taurine 110-60-1, Putrescine 110-86-1D, Pyridine, aminoalkyl derivs. 119-13-1, δ-Tocopherol 124-20-9, Spermidine 127-40-2, Lutein 144-68-3, Zeaxanthin 148-03-8, β-Tocopherol 149-91-7, Gallic acid, biological studies 153-18-4D, Rutoside, derivs. 404-86-4, Capsaicin 432-70-2, α-Carotene 462-20-4, Dihydrolipoic acid 462-94-2, Cadaverine 472-61-7, Astaxanthin 472-70-8, β-Cryptoxanthin 472-93-5, γ-Carotene 476-66-4, Ellagic acid 490-23-3, β-Tocotrienol 490-83-5, Dehydroascorbic acid 502-65-8, Lycopene 987-78-0, Citicoline 1200-22-2, α-Lipoic acid 1721-51-3, α-Tocotrienol 2078-54-8, Propofol 3376-24-7, α-Phenyl-N-tert-butyl nitrone 4671-06-1, Δ2-1,2,3-Triazoline 6829-55-6, Tocotrienol 7235-40-7, β-Carotene 7439-95-4, Magnesium, biological studies 7440-66-6, Zinc, biological studies 7616-22-0, γ-Tocopherol 7782-49-2, Selenium, biological studies 9000-94-6, Antithrombin III 9000-95-7, Apyrase 9001-05-2, Catalase 9004-54-0, Dextran, biological studies 9004-54-0D, Dextran, Hb conjugates 9005-27-0, Hydroxyethyl starch 9005-27-0D, Hydroxyethyl starch, Hb conjugates 9005-79-2, Glycogen, biological studies 9005-79-2D, Glycogen, Hb conjugates 9013-66-5, Glutathione peroxidase 9054-89-1, Superoxide dismutase 9055-67-8, Poly(ADP-ribose) polymerase 10540-29-1, Tamoxifen 10540-29-1D, Tamoxifen, metabolites 14101-61-2, γ-Tocotrienol 25322-68-3D, PEG, albumin reaction products 25612-59-3, δ-Tocotrienol 26993-30-6, Sphingosine-1-phosphate 29656-58-4D, Hydroxybenzoic acid, derivs. 57828-26-9, Lipoic acid 61805-96-7, Dimethylthiourea 64156-26-9 68047-06-3, 4-Hydroxytamoxifen 79902-63-9, Simvastatin

85637-73-6, Atrial natriuretic peptide 109319-16-6, Von Willebrand factor 154835-90-2, Adrenomedullin 168021-79-2, Cerovive 214210-47-6, Neuropilin 1
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(biodegradable macromols. with antioxidants and/or other chems. for capillary membrane stabilization and reduction of tissue injury)

IT 9002-17-9 9039-48-9, Aromatase 122320-05-2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; biodegradable macromols. with antioxidants and/or other chems. for capillary membrane stabilization and reduction of tissue injury)

L6 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:582000 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 145:40306
TITLE: Compositions and methods using polysaccharides and activated protein C for preventing and treating sepsis and other conditions
INVENTOR(S): Zikria, Bashir A.; Zikria, J. Dean
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 6 pp., Cont.-in-part of U.S. Ser. No. 837,840.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2006127387	A1	20060615	US 2005-280104		20051117 <--
US 7041655	B1	20060509	US 1997-837840		19970422 <--
PRIORITY APPLN. INFO.:			US 1997-837840	A2	19970422 <--
			US 1996-15963P	P	19960424 <--

AB The invention provides pharmaceutical compns. useful for the prevention and treatment of sepsis and other conditions, e.g. stroke, reperfusion injury, and heart attacks, containing (1) at least one macromol. polysaccharide selected from hydroxyethyl starch, dextran, glycogen, and mixts. thereof; and (2) activated protein C. The compns. can further comprise at least one member selected from the group consisting of at least one antioxidant and/or at least one antiinfective. The invention further provides methods for treating human subjects prior to or during sepsis and other conditions, e.g.stroke, reperfusion injury, and heart attacks to prevent leakage of macromols. from capillary endothelial junctions and simultaneously prevent thrombosis and fibrin formation, reduce inflammation and improve microcirculation, by i.v. administration of an effective amount of the composition

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2006127387	A1	20060615	US 2005-280104	20051117 <--
US 7041655	B1	20060509	US 1997-837840	19970422 <--
PRAI US 1997-837840	A2	19970422 <--		
US 1996-15963P	P	19960424 <--		

IT 9002-17-9, Xanthine oxidase 9039-48-9, Aromatase 9055-67-8, Poly(ADP-ribose) polymerase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; therapeutic compns. and methods using polysaccharides and activated protein C)

IT 50-81-7, Vitamin C, biological studies 51-45-6, Histamine, biological studies 56-65-5, Adenosine triphosphate, biological studies 58-61-7, Adenosine, biological studies 58-63-9, Inosine 60-92-4 73-31-4, Melatonin 89-25-8, Edaravone 107-35-7, Taurine 110-86-1D, Pyridine, aminoalkyl derivs. 153-18-4D, Rutoside, derivs. 490-83-5, Dehydroascorbic acid 987-78-0, Citicoline 2078-54-8, Propofol 3376-24-7, α-Phenyl-N-tert-butyl nitrone 4671-06-1, Δ2-1,2,3-Triazoline 6829-55-6, Tocotrienol 9000-94-6, Antithrombin III 9000-95-7, Apyrase 9001-05-2, Catalase 9004-54-0, Dextran, biological studies 9005-27-0, Hydroxyethyl starch 9005-79-2, Glycogen, biological studies 9013-66-5, Glutathione peroxidase 9054-89-1, Superoxide dismutase 10540-29-1, Tamoxifen 10540-29-1D, Tamoxifen, metabolites 11030-71-0, Amanitin 17466-45-4, Phalloidin 25322-68-3D, PEG, albumin conjugates 26993-30-6, Sphingosine-1-phosphate 42617-41-4, Activated protein C 57828-26-9, Lipoic acid 61805-96-7, Dimethylthiourea 64156-26-9 79902-63-9, Simvastatin 85637-73-6, Atrial natriuretic peptide 109319-16-6, Von Willebrand factor 122320-05-2, Secretory leukocyte protease inhibitor 154835-90-2, Adrenomedullin 168021-79-2, Cerovive 214210-47-6, Neuropilin 1
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(therapeutic compns. and methods using polysaccharides and activated protein C)

L6 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:498003 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 135:254973
TITLE: Vitamin C Transport in Human Lens Epithelial Cells: Evidence for the Presence of SVCT2
AUTHOR(S): Kannan, R.; Stolz, A.; Ji, Q.; Prasad, P. D.; Ganapathy, V.
CORPORATE SOURCE: USC Keck School of Medicine, Los Angeles, CA, 90033, USA
SOURCE: Experimental Eye Research (2001), 73(2), 159-165
CODEN: EXERA6; ISSN: 0014-4835
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Vitamin C [ascorbic acid (AA)] is an important antioxidant present in mM amts. in the aqueous humor. Recently, two specific transporters for vitamin C (SVCT1, SVCT2) have been cloned in the rat and the human. The aim of the present study was to characterize vitamin C transport in an immortalized human lens epithelial cell line (HLE-B3). AA uptake was linear for 120 min in expts. conducted with 14C AA + 40 μM unlabeled AA. Uptake was measured at varying AA concns. (0.04-1 m M) in Na+-containing and Na+-free buffers for 30 min at 37°C. Effect of potential inhibitors of AA transport was also examined Presence (or absence) of SVCT1 and SVCT2 was studied by RT-PCR of HLE-B3 poly(A)+ RNA using gene specific primers. Uptake studies revealed that AA uptake was highly Na+-dependent and exhibited saturation Na+-dependent 14C-AA uptake was strongly inhibited (85-90%) by 10 mM unlabeled AA. Incubation of HLE-B3 cells with cAMP (0.1 mM), cytochalasin B (0.1 m M) and phorbol dibutyrate (1 μM) resulted in partial inhibition (36-51%) of AA uptake. Under similar conditions, D-glucose (10 mM) and staurosporine (0.1 μM) had no effect. RT-PCR showed the presence of SVCT2 while SVCT1 could not be amplified. Exposure to the chemical oxidant tert-butylhydroperoxide (TBH) up-regulated SVCT2 gene

expression in HLE-B3 cells. Our data suggest that Na+-dependent transport of AA in normal lens epithelium is most likely mediated by SVCT2 rather than by SVCT1. This transport system may be subject to regulation by oxidant stress and by various second messenger signals. (c) 2001 Academic Press.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Vitamin C Transport in Human Lens Epithelial Cells: Evidence for the Presence of SVCT2

SO Experimental Eye Research (2001), 73(2), 159-165
CODEN: EXERA6; ISSN: 0014-4835

AB Vitamin C [ascorbic acid (AA)] is an important antioxidant present in mM amts. in the aqueous humor. Recently, two specific transporters for vitamin C (SVCT1, SVCT2) have been cloned in the rat and the human. The aim of the present study was to characterize vitamin C transport in an immortalized human lens epithelial cell line (HLE-B3). AA uptake was linear for 120 min in expts. conducted. . . at varying AA concns. (0.04-1 m M) in Na+-containing and Na+-free buffers for 30 min at 37°C. Effect of potential inhibitors of AA transport was also examined Presence (or absence) of SVCT1 and SVCT2 was studied by RT-PCR of HLE-B3 poly(A)+. . .

ST vitamin C transport SVCT2 transporter lens epithelium oxidative stress; ascorbate transport SVCT2 sodium cAMP protein kinase C lens

IT Oxidative stress, biological
(SVCT2 transporter regulated by oxidative stress and by second messenger signals in vitamin C transport in human lens epithelial cells)

IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(SVCT2; SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT Transport proteins
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(ascorbate-sodium-cotransporting, SVCT2 (sodium-vitamin C-transporting, 2); SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT Eye
(lens, epithelium; SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT Biological transport
(uptake, carrier-mediated; SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT 7440-23-5, Sodium, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT 50-81-7, Vitamin C, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(SVCT2 transporter in vitamin C transport in human lens epithelial cells)

IT 141436-78-4, Protein kinase C

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(SVCT2 transporter regulated by oxidative stress and by second messenger signals in vitamin C transport in human lens epithelial cells)

IT 60-92-4, CAMP
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(SVCT2 transporter regulated by oxidative stress and by second messenger signals in vitamin C transport in human lens epithelial cells)

L6 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:585381 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 133:182770
TITLE: Antiaging cosmetics containing tomato pigments
INVENTOR(S): Uehara, Shizuka; Kameyama, Kumi; Kondo, Chiharu; Takada, Norihisa
PATENT ASSIGNEE(S): Kosei Co., Ltd., Japan; Nippon Delmonte K. K.
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000229827	A	20000822	JP 1999-28301	19990205 <--

PRIORITY APPLN. INFO.: JP 1999-28301 19990205 <--
AB The cosmetics are claimed. The tomato pigments may mainly comprise lycopene isolated by centrifugation of tomato prepns., microfiltration of the liquid parts, and collection of unfiltered substances by microfiltration. The cosmetics may addnl. contain active oxygen scavengers, antioxidants, inflammation inhibitors, UV shields, cell activators, and/or moisturizers. A cream containing the tomato pigment was used by volunteers to lighten skin and increase elasticity.
PI JP 2000229827 A 20000822

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2000229827	A	20000822	JP 1999-28301	19990205 <--

PRAI JP 1999-28301 19990205 <--
AB . . . the liquid parts, and collection of unfiltered substances by microfiltration. The cosmetics may addnl. contain active oxygen scavengers, antioxidants, inflammation inhibitors, UV shields, cell activators, and/or moisturizers. A cream containing the tomato pigment was used by volunteers to lighten skin and. . .
IT 50-81-7, Vitamin C, biological studies 59-43-8, biological studies 1406-16-2, Vitamin D 1406-18-4, Vitamin E 11103-57-4, Vitamin A 30587-81-6, Dibutylhydroxytoluene 82321-68-4, Dibutylhydroxyanisole
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(antioxidant; antiaging cosmetics containing tomato pigments mainly comprising lycopene complexes and other active ingredients)
IT 50-21-5, biological studies 50-28-2, Estradiol, biological studies 50-70-4, Sorbitol, biological studies 50-99-7, Glucose, biological studies 51-35-4, Hydroxyproline 52-90-4, Cysteine, biological studies

56-40-6, Glycine, biological studies 56-41-7, L-Alanine, biological studies 56-45-1, Serine, biological studies 56-65-5, Adenosine triphosphate, biological studies 56-84-8, Aspartic acid, biological studies 56-85-9, Glutamine, biological studies 56-86-0, Glutamic acid, biological studies 56-87-1, Lysine, biological studies 56-89-3, Cystine, biological studies 57-13-6, Urea, biological studies 57-48-7, Fructose, biological studies 57-50-1, biological studies 58-08-2, Caffeine, biological studies 58-55-9, Theophylline, biological studies 58-64-0, Adenosine diphosphate, biological studies 58-86-6, Xylose, biological studies 60-18-4, Tyrosine, biological studies 60-92-4 61-19-8, Adenosine monophosphate, biological studies 63-68-3, Methionine, biological studies 63-91-2, Phenylalanine, biological studies 65-71-4, Thymine 69-72-7, biological studies 69-79-4, Maltose 69-89-6, Xanthine 70-26-8, Ornithine 70-47-3, Asparagine, biological studies 71-30-7, Cytosine 72-18-4, Valine, biological studies 72-19-5, Threonine, biological studies 73-24-5, Adenine, biological studies 73-32-5, Isoleucine, biological studies 73-40-5, Guanine 74-79-3, Arginine, biological studies 77-92-9, biological studies 79-14-1, biological studies 81-13-0, D-Panthenol 87-69-4, biological studies 87-89-8, Inositol 87-99-0, Xylitol 98-79-3, Pyrrolidonecarboxylic acid 99-20-7, Trehalose 110-15-6, Butanedioic acid, biological studies 115-77-5, biological studies 146-14-5, Flavin adenine dinucleotide 147-85-3, Proline, biological studies 149-32-6, Erythritol 372-75-8, Citrulline 463-40-1, α-Linolenic acid 481-49-2, Cepharanthine 499-44-5, Hinokitiol 506-26-3, γ-Linolenic acid 585-88-6, Maltitol 1190-94-9, Hydroxylysine 3081-61-6, Theanine 6915-15-7 7665-99-8, Cyclic GMP 7678-95-7 9004-53-9, Dextrin 9004-61-9, Hyaluronic acid 9005-49-6, Heparin, biological studies 9007-28-7, Chondroitin sulfate 9050-30-0, Heparan sulfate 9056-36-4, Keratan sulfate 24967-94-0, Dermatan sulfate 25378-27-2, Eicosapentaenoic acid
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(cell activator; antiaging cosmetics containing tomato pigments mainly comprising lycopene complexes and other active ingredients)

IT 50-33-9, Phenylbutazone, biological studies 53-86-1, Indomethacin 60-32-2 61-68-7, Mefenamic acid 97-59-6, Allantoin 471-53-4, Glycyrrhetinic acid 489-84-9, Guaiazulene 1197-18-8, Tranexamic acid 1405-86-3, Glycyrrhizinic acid 15307-79-6, Diclofenac sodium 15687-27-1, Ibuprofen 22071-15-4, Ketoprofen
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(inflammation inhibitor; antiaging cosmetics containing tomato pigments mainly comprising lycopene complexes and other active ingredients)

L6 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2000:69961 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 133:334
TITLE: Induction of cell death by ascorbic acid derivatives in human renal carcinoma and glioblastoma cell lines
AUTHOR(S): Makino, Yasushi; Sakagami, Hiroshi; Takeda, Minoru
CORPORATE SOURCE: First Department of Biochemistry, School of Medicine, Showa University, Tokyo, 142-8555, Japan
SOURCE: Anticancer Research (1999), 19(4B), 3125-3132
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Sodium-L-ascorbate, L-ascorbic acid, D-isoascorbic acid, sodium 5,6-benzylidene-L-ascorbate and sodium-6-β-O-galactosyl-L-ascorbate, which produce ascorbyl radicals during the oxidative degradation, also induced cytotoxicity against cultured human renal carcinoma (TC-1) and glioblastoma multiform tumor (T98G) cell lines. On the other hand, L-ascorbic acid 2-phosphate magnesium and L-ascorbic acid 2-sulfate dipotassium salt, which do not produce the ascorbyl radical, were inactive. This suggests the possible role of the ascorbyl radical for cell death induction. T98G cells were more resistant to ascorbate analogs than TC-1 and HL-60 cells, possibly due to higher intracellular glutathione concns. Ascorbate treatment induced rapid elevation of both intracellular concentration of cAMP and Ca2+ in HL-60 cells, but not in TC-1 and T98G cells. However, the elevation of cAMP by theophylline and N,2-dibutyryl adenosine 3,5 cyclic monophosphate (dibutyryl cAMP) resulted in a decrease in the viable cell number This suggests the possible role of cAMP for ascorbate-induced cell death.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO Anticancer Research (1999), 19(4B), 3125-3132
CODEN: ANTRD4; ISSN: 0250-7005

AB Sodium-L-ascorbate, L-ascorbic acid, D-isoascorbic acid, sodium 5,6-benzylidene-L-ascorbate and sodium-6-β-O-galactosyl-L-ascorbate, which produce ascorbyl radicals during the oxidative degradation, also induced cytotoxicity against cultured human renal carcinoma (TC-1) and glioblastoma multiform tumor (T98G) cell lines. On the other hand, L-ascorbic acid 2-phosphate magnesium and L-ascorbic acid 2-sulfate dipotassium salt, which do not produce the ascorbyl radical, were inactive. This suggests the possible role of the ascorbyl. . .

IT Kidney, neoplasm
(carcinoma, inhibitors; induction of cell death by ascorbic acid derivs. in human renal carcinoma and glioblastoma cell lines)

IT Neuroglia
Neuroglia
(glioblastoma, inhibitors; induction of cell death by ascorbic acid derivs. in human renal carcinoma and glioblastoma cell lines)

IT Kidney, neoplasm
(renal cell carcinoma, inhibitors; induction of cell death by ascorbic acid derivs. in human renal carcinoma and glioblastoma cell lines)

IT 50-81-7, L-Ascorbic acid, biological studies
50-81-7D, L-Ascorbic acid, derivs., biological studies 89-65-6, D-Isoascorbic acid 134-03-2, Sodium-L-ascorbate 490-83-5, Dehydroascorbic acid 23666-04-8 52174-99-9 98734-55-5, Sodium 5,6-benzylidene-L-ascorbate 136521-47-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(induction of cell death by ascorbic acid derivs. in human renal carcinoma and glioblastoma cell lines)

IT 60-92-4 6730-29-6, Ascorbyl radical, biological studies
14127-61-8, Ca2+, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(induction of cell death by ascorbic acid derivs. in human renal carcinoma and glioblastoma cell lines)

=> FIL STNGUIDE
COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION
FULL ESTIMATED COST | 35.82 | 38.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION
CA SUBSCRIBER PRICE | -3.90 | -3.90

FILE 'STNGUIDE' ENTERED AT 11:21:35 ON 19 NOV 2007

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Nov 16, 2007 (20071116/UP).

=> d 7-14 ibib abs kwic
YOU HAVE REQUESTED DATA FROM FILE 'CAPLUS' - CONTINUE? (Y)/N:y

L6 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:206633 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 128:304304
TITLE: The effect of prostaglandin E2 on costochondral chondrocyte differentiation is mediated by cyclic adenosine 3',5'-monophosphate and protein kinase C
AUTHOR(S): Schwartz, Z.; Gilley, R. M.; Sylvia, V. L.; Dean, D. D.; Boyan, B. D.
CORPORATE SOURCE: Department of Periodontics, University of Texas Health Science Center, San Antonio, TX, 78284, USA
SOURCE: Endocrinology (1998), 139(4), 1825-1834
CODEN: ENDOAO; ISSN: 0013-7227
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Recent studies indicate that vitamin D metabolites exert rapid effects on growth plate chondrocytes via changes in PG production and protein kinase C (PKC) activity. This suggests that these two products of vitamin D action may be interrelated. To test this hypothesis, the authors examined the effect of PGE2 on rat costochondral resting zone and growth zone cartilage cells and determined whether the effects of PGE2 are mediated by changes in the level of cAMP and/or PKC activity, whether there is a relationship between cAMP production and PKC activity, and whether cell maturation-specific effects are involved. Confluent, fourth passage resting zone and growth zone cartilage cell cultures were incubated in DMEM containing 10% FBS, 50 μg/mL vitamin C, and 1% antibiotics. The PGE2 concentration was varied from 0.007-15 ng/mL. Low concns. of PGE2 caused a dose-dependent increase in cell number and [3H]thymidine incorporation and stimulated alkaline phosphatase specific activity. These effects were comparable in resting zone and growth zone cartilage cells at the same PGE2 concns. At higher concns., PGE2 caused a general increase in the synthesis of collagenase-digestible protein and noncollagenase-digestible protein in resting zone cartilage cells and of collagenase-digestible protein in growth zone cartilage cells, resulting in a net increase in the percent collagen synthesis for both cell types. The cAMP production was increased over the entire range of chondrocyte response. Prevention of cAMP metabolism

with the protein kinase A inhibitors H-8 and H-89 blocked the PGE2-dependent inhibition of PKC in resting zone cartilage cells in a dose-dependent manner. H-8 alone had no effect on PKC in resting zone cartilage cells, but stimulated PKC activity in growth zone cartilage cells; H-89 alone stimulated PKC activity in resting zone cartilage cells. These results suggest that low levels of PGE2 promote differentiation, whereas high doses promote an anabolic response; PGE2 increases cAMP production and PKC activity in a cell maturation-dependent manner; PGE2 exerts its effects via cAMP production and PKC activity; and regulation of PGE2-dependent PKC is via cAMP.

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO Endocrinology (1998), 139(4), 1825-1834
CODEN: ENDOAO; ISSN: 0013-7227

AB . . . Confluent, fourth passage resting zone and growth zone cartilage cell cultures were incubated in DMEM containing 10% FBS, 50 μg/mL vitamin C, and 1% antibiotics. The PGE2 concentration was varied from 0.007-15 ng/mL. Low concns. of PGE2 caused a dose-dependent increase in. . . cAMP production was increased over the entire range of chondrocyte response. Prevention of cAMP metabolism with the protein kinase A inhibitors H-8 and H-89 blocked the PGE2-dependent inhibition of PKC in resting zone cartilage cells in a dose-dependent manner. H-8 alone. . .

IT 60-92-4, CAMP 141436-78-4, Protein kinase C
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(PGE2 effects on costochondral chondrocyte differentiation mediation by cAMP and protein kinase C)

L6 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:756973 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 128:39400
TITLE: Topical slimming composition containing plant extracts
INVENTOR(S): Bonte, Frederic; Meybeck, Alain
PATENT ASSIGNEE(S): Lvmh Recherche, Fr.; Bonte, Frederic; Meybeck, Alain
SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9742928	A1	19971120	WO 1997-IB553	19970514 <--
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
FR 2748659	A1	19971121	FR 1996-5968	19960514 <--
FR 2748659	B1	19980724		

PRIORITY APPLN. INFO.: FR 1996-5968 A 19960514 <--

AB A cosmetic or pharmaceutical slimming composition comprises an aqueous phase which is preferably non alc., and a hydro-alc. phase. Each of these two phases comprises at least one active substance which stimulates the lipolysis, and the microcirculation or which inhibits the cutaneous inflammatory process, said substance being compatible with other compds. of the phase where it is incorporated. Said phases are conditioned sep. from each other but are used in simultaneous application on the skin. Thus, the invention enables to avoid the problems due to incompatibilities of

formulation, while preserving the activity of the composition and its comfort of use. The slimming compns. of the invention may be used in topical application on the various parts of the body where a local reduction of fat is desired, particularly the s.c. fatty tissues, as well as to reduce the risks of forming vibices. Cosmetic compns. for slimming contained caffeine 0.1-2, horse chestnut extract 0.2, horsetail extract 3, St. John's wort extract, echinacea extract 3, ammonium glycyrrhizinate 0.2, cAMP 0.005-0.02, Carbopol ETD 2020 0.1-1, propylene glycol 0.2-2, and water q.s. 100 g in the hydroalcoholic phase and ruscogenine 0.1-0.2, green tea ext 0.1, Ginkgo biloba 0.2, glycerol 0.1-2, malic acid 0.1, Crbopol ETD 2020 0.5-1, 95% ethanol 37-68, and fragrance, preservative and water q.s. 100 g in the hydro-alc. phase.

PI WO 9742928 A1 19971120

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9742928	A1	19971120	WO 1997-IB553	19970514 <--
W: JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
FR 2748659	A1	19971121	FR 1996-5968	19960514 <--
FR 2748659	B1	19980724		

PRAI FR 1996-5968 A 19960514 <--

IT 69-89-6
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; topical slimming composition containing plant exts.)

IT 50-81-7, L-Ascorbic acid, biological studies
56-81-5, 1,2,3-Propanetriol, biological studies 58-08-2, biological studies 58-55-9, biological studies 60-92-4 64-17-5, Ethanol, biological studies 67-63-0, 2-Propanol, biological studies 71-23-8, 1-Propanol, biological studies 471-53-4 471-53-4D, esters 472-11-7 477-32-7 541-15-1 6915-15-7 53956-04-0 55306-04-2 68797-35-3 176429-87-1, Carbopol ETD 2020
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(topical slimming composition containing plant exts.)

L6 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:290554 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 126:297470
TITLE: Use of Eriobotrya japonica extract in cosmetics for stimulating glycosaminoglycan synthesis
INVENTOR(S): Bonte, Frederic; Dumas, Marc
PATENT ASSIGNEE(S): Lvmn Recherche, Fr.
SOURCE: PCT Int. Appl., 16 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9706659	A2	19970227	WO 1997-FR9	19970103 <--
WO 9706659	A3	19971023		
W: AT, AU, CA, CH, CN, CU, CZ, DE, DK, ES, FI, GB, HU, IL, JP, LU, NO, NZ, PL, PT, RO, RU, UA, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, NL, PT, SE				
FR 2742987	A1	19970704	FR 1996-18	19960103 <--
FR 2742987	B1	19980403		

AU 9711810	A	19970312	AU 1997-11810		19970103 <--
BE 1010042	A3	19971202	BE 1997-1		19970103 <--
GB 2314272	A	19971224	GB 1997-18684		19970103 <--
DE 19780092	T0	19980226	DE 1997-19780092		19970103 <--
JP 11501325	T	19990202	JP 1997-508997		19970103 <--
CH 692902	A5	20021213	CH 1997-2066		19970103 <--
NL 1004939	C2	19970707	NL 1997-1004939		19970106 <--
NL 1004939	A1	19970707			
ES 2129014	A1	19990516	ES 1997-50020		19970903 <--
ES 2129014	B1	20000216			
PRIORITY APPLN. INFO.:			FR 1996-18	A	19960103 <--
			WO 1997-FR9	W	19970103 <--

AB The invention concerns novel uses of an Eriobotrya japonica extract, in particular in cosmetics of pharmaceuticals. This extract permits stimulation of glycosaminoglycan synthesis, in particular of hyaluronic acid, thus imparting to the cosmetic compns. containing this extract the properties of improving the firmness and suppleness of the skin, combating the formation of wrinkles or lessening the depth thereof, smoothing the surface of the skin by means of a tightening effect, or moisturizing the skin. The invention further concerns the use of these exts. for stimulating the synthesis of glycosaminoglycans from a cell culture medium, in particular fibroblasts or keratinocytes. Dried leaves of E. japonica was extracted with 20 mL of a 50:50 mixture of 1,3-butylene glycol and water at 35° for 1 h, the suspension thus obtained was then filtered. The extract (10 μg/mL) increased the glycosaminoglycans produced by cultured human fibroblast by 83%. A cosmetic gel contained the above extract 3, ascorbic acid magnesium phosphate salt 1, asiaticoside 0.1, and excipients q.s. 100 g.

PI WO 9706659 A2 19970227

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9706659	A2	19970227	WO 1997-FR9	19970103 <--
WO 9706659	A3	19971023		
W: AT, AU, CA, CH, CN, CU, CZ, DE, DK, ES, FI, GB, HU, IL, JP, LU, NO, NZ, PL, PT, RO, RU, UA, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, NL, PT, SE				
FR 2742987	A1	19970704	FR 1996-18	19960103 <--
FR 2742987	B1	19980403		
AU 9711810	A	19970312	AU 1997-11810	19970103 <--
BE 1010042	A3	19971202	BE 1997-1	19970103 <--
GB 2314272	A	19971224	GB 1997-18684	19970103 <--
DE 19780092	T0	19980226	DE 1997-19780092	19970103 <--
JP 11501325	T	19990202	JP 1997-508997	19970103 <--
CH 692902	A5	20021213	CH 1997-2066	19970103 <--
NL 1004939	C2	19970707	NL 1997-1004939	19970106 <--
NL 1004939	A1	19970707		
ES 2129014	A1	19990516	ES 1997-50020	19970903 <--
ES 2129014	B1	20000216		
PRAI FR 1996-18	A	19960103 <--		
WO 1997-FR9	W	19970103 <--		

IT 9001-54-1, Hyaluronidase 9004-06-2, Elastase 9025-82-5, Phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; use of Eriobotrya japonica extract in cosmetics for stimulating glycosaminoglycan synthesis)

IT 60-92-4, Cyclic amp
RL: BSU (Biological study, unclassified); BIOL (Biological study) (stimulants of synthesis of; use of Eriobotrya japonica extract in cosmetics for stimulating glycosaminoglycan synthesis)

IT 50-81-7, Vitamin c, biological studies 51-35-4, Hydroxyproline 56-45-1, L-Serine, biological studies 56-81-5, 1,2,3-Propanetriol, biological studies 58-08-2, Caffein, biological studies 58-55-9, Theophylline, biological studies 61-90-5, Leucine, biological studies 68-19-9, Vitamin b 12 68-26-8, Retinol 72-19-5, Threonine, biological studies 79-81-2, Retinol palmitate 116-31-4, Retinaldehyde 127-47-9, Retinol acetate 134-03-2, Sodium ascorbate 147-85-3, Proline, biological studies 302-79-4, Retinoic acid 464-92-6, Asiatic acid 1406-18-4, Vitamin e 3416-24-8, Glucosamine 7069-42-3, Retinol propionate 7535-00-4, Galactosamine 8059-24-3, Vitamin b 6 11032-50-1, Vitamin pp 11103-57-4, Vitamin a 12001-76-2, Vitamin b complex 15431-40-0, Magnesium ascorbate 16830-15-2, Asiaticoside 18449-41-7, Madecassic acid 25322-68-3 34540-22-2, Madecassoside 66575-29-9, Forskolin
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of Eriobotrya japonica extract in cosmetics for stimulating glycosaminoglycan synthesis)

L6 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1996:577828 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 125:269861
TITLE: Solution for prolonged organ preservation
INVENTOR(S): Stern, David M.; Oz, Mehmet C.; Nowygrod, Roman; Koga, Shin; Pinsky, David J.
PATENT ASSIGNEE(S): The Trustees of Columbia University In the City of New York, USA
SOURCE: U.S., 71 pp., Cont.-in-part of U.S. 5,370,989.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5552267	A	19960903	US 1994-350319	19941205 <--
US 5370989	A	19941206	US 1994-206197	19940303 <--
PRIORITY APPLN. INFO.:			US 1992-863197	B1 19920403 <--
			US 1994-206197	A2 19940303 <--

AB An aqueous solution for organ preservation or maintenance contains: a vasodilator in an amount sufficient to maintain vascular homeostasis; D-glucose and Mg2+ in amts. sufficient to support intracellular function and maintenance of cellular bioenergetics; macromols. of mol. weight >20,000 in an amount sufficient to maintain endothelial integrity and cellular viability; >100 mM K+; and a buffer in an amount sufficient to maintain the average pH of the organ preservation or maintenance solution during the period of organ preservation at or above physiol. pH. A suitable solution for heart preservation (Columbia University solution) contained D-glucose 67.4, MgSO4 5, K gluconate 95, adenosine 5, N-acetylcysteine 0.5, dibutyryl cAMP 2, KH2PO4 25 mM, heparin 10 U/mL, dextran 50 g/L, cefazolin 0.5, nitroglycerin 0.1 mg/mL, verapamil 10, BHA 50, and BHT 50 μM. Restoration of the cAMP 2nd messenger pathway, and supplementation of the NO pathway with nitroglycerin, nitroprusside, or L-arginine, enhanced cardiac preservation for transplantation in a heterotopic rat model. The NO/cGMP pathway also had a critical role in successful lung preservation.
PI US 5552267 A 19960903

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI	US 5552267	A	19960903	US 1994-350319	19941205 <--
	US 5370989	A	19941206	US 1994-206197	19940303 <--
PRAI	US 1992-863197	B1	19920403 <--		
	US 1994-206197	A2	19940303 <--		

IT Blood platelet aggregation inhibitors
(nitroglycerin; solution for prolonged organ preservation)
IT 9036-21-9, CAMP phosphodiesterase 9068-52-4, CGMP phosphodiesterase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; solution for prolonged organ preservation)
IT 60-92-4, CAMP
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(of vascular smooth muscle, hypoxia effect on)
IT 50-81-7, Vitamin C, biological studies 50-99-7, D-Glucose, biological studies 52-53-9, Verapamil 55-63-0, Nitroglycerin 58-61-7, Adenosine, biological studies 60-92-4D, CAMP, analogs 74-79-3, Arginine, biological studies 96-82-2, Lactobionic acid 128-37-0, BHT, biological studies 299-27-4, Potassium gluconate 362-74-3, Dibutyryl cAMP 526-95-4, Gluconic acid 616-91-1, N-Acetylcysteine 1406-05-9, Penicillin 1406-18-4, Vitamin E 3632-91-5, Magnesium gluconate 7439-95-4, Magnesium, biological studies 7440-09-7, Potassium, biological studies 7487-88-9, Magnesium sulfate, biological studies 7665-99-8D, CGMP, analogs 7778-77-0, Monopotassium phosphate 7778-80-5, Potassium sulfate, biological studies 8001-27-2, Hirudin 9004-54-0, Dextran, biological studies 9005-49-6, Heparin, biological studies 9054-89-1, Superoxide dismutase 10043-83-1 15078-28-1, Nitroprusside 25013-16-5 25322-68-3 25953-19-9, Cefazolin 28822-58-4, IBMX 31356-94-2, 8-Bromo-cGMP 37762-06-4 61413-54-5, Rolipram 100643-96-7, Indolidan
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(solution for prolonged organ preservation)

L6 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER:	1995:644046 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER:	123:52884
TITLE:	Ascorbate transport and intracellular concentration in cerebral astrocytes
AUTHOR(S):	Siushansian, Ramin; Wilson, John X.
CORPORATE SOURCE:	Department of Physiology, University of Western Ontario, London, ON, Can.
SOURCE:	Journal of Neurochemistry (1995), 65(1), 41-9 CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER:	Raven
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Regulation of the initial rate of uptake and steady-state concentration of ascorbate (reduced vitamin C) was investigated in rat cerebral astrocytes. Although these cells did not synthesize vitamin C, they accumulated millimolar concns. of ascorbate when incubated with medium containing the vitamin at a level (200 μM) typical of brain extracellular fluid. Initial rate of [14C]-ascorbate uptake and intracellular ascorbate concentration were dependent on extracellular Na+ and sensitive to the anion transport inhibitor sulfinpyrazone. Comparison of the efflux profiles of ascorbate and 2',7'-bis(carboxyethyl)-5 (or -6)-carboxyfluorescein from

astrocytes permeabilized with digitonin localized most intracellular ascorbate to the cytosol. Pretreatment of astrocytes with dibutyryl cAMP (dBcAMP) doubled their initial rate of sulfinpyrazone-sensitive [14C]ascorbate uptake compared with cells treated with either n-butyric acid or vehicle. DBcAMP also increased steady-state intracellular ascorbate concentration at 39%. The relatively small size of the change in astrocytic ascorbate concentration was explained by the finding that dBcAMP increased the rate of efflux of the vitamin for ascorbate-loaded cells. These results indicate that uptake and efflux pathways are stimulated by cAMP-dependent mechanisms and that they regulate the cytosolic concentration of ascorbate in astrocytes.

SO Journal of Neurochemistry (1995), 65(1), 41-9
CODEN: JONRA9; ISSN: 0022-3042

AB Regulation of the initial rate of uptake and steady-state concentration of ascorbate (reduced vitamin C) was investigated in rat cerebral astrocytes. Although these cells did not synthesize vitamin C, they accumulated millimolar concns. of ascorbate when incubated with medium containing the vitamin at a level (200 μM) typical of. . . Initial rate of [14C]-ascorbate uptake and intracellular ascorbate concentration were dependent on extracellular Na+ and sensitive to the anion transport inhibitor sulfinpyrazone. Comparison of the efflux profiles of ascorbate and 2',7'-bis(carboxyethyl)-5 (or -6)-carboxyfluorescein from astrocytes permeabilized with digitonin localized most intracellular. . .

IT 60-92-4, Cyclic AMP
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(ascorbate transport by cerebral astrocytes regulation by)

L6 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1985:201411 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 102:201411
TITLE: Developmental physiology of cestodes: cyclic nucleotides and the identity of putative crowding factors in Hymenolepis diminuta
AUTHOR(S): Zavras, Eugenia T.; Roberts, Larry S.
CORPORATE SOURCE: Dep. Biol. Sci., Texas Tech Univ., Lubbock, TX, 79409, USA
SOURCE: Journal of Parasitology (1985), 71(1), 96-105
CODEN: JOPAA2; ISSN: 0022-3395
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Worm-conditioned saline (WCS) was prepared by incubating H. diminuta from crowded infections for 12 h in a balanced salt solution The effect of the WCS on the incorporation of [3H]thymidine into DNA in the anterior regions of fresh H. diminuta was compared to effects produced by the cyclic nucleotides in the WCS. Cyclic AMP and cGMP were found in the WCS, and cGMP, but not cAMP (at the concentration in WCS), caused some inhibition of DNA synthesis. Worms were incubated with theophylline, caffeine, IBMX, 2-deoxy cGMP, and L-ascorbic acid, all of which produced some inhibition of [3H]thymidine incorporation. Treatment of WCS with 3',5'-cyclic nucleotide phosphodiesterase abolished part of its inhibitory activity, i.e., that part presumed to be due to cGMP. When worms were incubated in the presence of succinic acid, acetic acid, D-glucosaminic acid, and cGMP simultaneously and in the concns. each was found in the WCS, DNA synthesis was inhibited to a degree equal to that found in the WCS. Thus these substances apparently represent the putative crowding factors in the WCS. WCS prepared with worms from different

population densities contained the same levels of cAMP but varied in content of cGMP, which decreased as the worm d. increased. WCS prepared with patent worms contained high levels of cAMP, but the same amts. of cGMP as WCS prepared with 10-day-old worms. At least some inhibitors of cyclic nucleotide phosphodiesterase inhibited the secretion of cGMP by the worms. Levels of cGMP in the host intestine varied with the presence or absence of worms, number of worms, and area of the intestine.

SO Journal of Parasitology (1985), 71(1), 96-105
CODEN: JOPAA2; ISSN: 0022-3395

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IT 60-92-4 64-19-7, biological studies 110-15-6, biological studies 3646-68-2 7665-99-8 32266-35-6
RL: BIOL (Biological study)
(DNA formation by tapeworm response to, crowding factors in relation to)

L6 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1984:400337 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 101:337
TITLE: Effect of hyperthermia in combination with vitamin E and cyclic AMP on neuroblastoma cells in culture
AUTHOR(S): Rama, Bhola N.; Prasad, Kedar N.
CORPORATE SOURCE: Sch. Med., Univ. Colorado, Denver, CO, 80262, USA
SOURCE: Life Sciences (1984), 34(21), 2089-97
CODEN: LIFSAK; ISSN: 0024-3205
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of heat in combination with DL-α-tocopheryl succinate (vitamin E succinate) [17407-37-3] and cAMP [60-92-4] stimulating agents on mouse neuroblastoma cells (NBP2) in culture on the criterion of growth inhibition (due to cell death and inhibition of cell division) was studied. Heat (41°-40°) alone inhibited growth; however, the extent of growth inhibition was dependent upon the temperature and the time of heat treatment. Heat (41°-40°) in combination with vitamin E succinate (5 μg/mL) produced an additive effect on the criterion of growth inhibition. Vitamin C [50-81-7] (100 μg/mL) failed to modify the effect of heat. Prostaglandin A2 [13345-50-1], a stimulator of adenylate cyclase, and 4-(3-butoxy-4-methoxybenzyl)-2-imidazolidinone (R020-1724) [29925-17-5], an inhibitor of cyclic nucleotide phosphodiesterase, are known to induce irreversible differentiation in mouse neuroblastoma cells in culture. These agents, in combination with heat (40°) produced a synergistic effect on the criterion of growth inhibition. Apparently the addition of vitamin E and cAMP stimulating agents may increase the effectiveness of hyperthermia protocol.

SO Life Sciences (1984), 34(21), 2089-97
CODEN: LIFSAK; ISSN: 0024-3205

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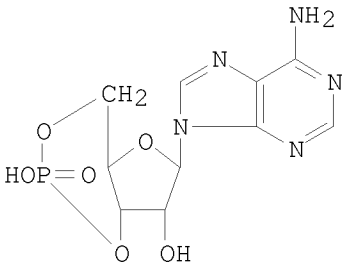
IT Neoplasm inhibitors
(cAMP and vitamin E as, in hyperthermia)

IT 60-92-4
RL: BIOL (Biological study)
(agents stimulating, neuroblastoma inhibition by heat and)

L6 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1983:174503 CAPLUS <<LOGINID::20071119>>
DOCUMENT NUMBER: 98:174503
TITLE: Use of cultures of neuroblastoma and glioma as a model system to study the heavy metal-induced neurotoxicity
AUTHOR(S): Prasad, Kedar N.
CORPORATE SOURCE: Med. Cent., Univ. Colorado, Denver, CO, 80262, USA
SOURCE: NATO Conference Series I: Ecology (1983), 5A(In Vitro Toxic. Test. Environ. Agents: Curr. Future Possibilities, Pt. A), 421-72
CODEN: NCSEDQ; ISSN: 0197-4475
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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AB Monolayer cultures of neuroblastoma (NB) and glioma cells were used as an exptl. model to study the cellular and mol. mechanisms of toxicity of heavy metals to nervous tissue. Glioma cells were more sensitive to MeHgCl [115-09-3] than NB for the criterion of growth inhibition. HgCl2, Bu3Pb(OAc) [2587-82-8], and acrylamide [79-06-1] did not produce such a differential effect. vitamin E [1406-18-4] And inhibitors of cyclic nucleotide phosphodiesterase (papaverine [58-74-2], R 020-1724 [29925-17-5], and isobutylxanthic acid [6791-12-4]) protected glioma cells against MeHgCl-induced toxicity; however, it did not protect NB cells. vitamin C [50-81-7] Enhanced the effect of MeHgCl on NB cells, but not on glioma cells. Glioma cells produce factor(s) in the medium which enhanced the effect of MeHgCl on glioma and NB cells. MeHgCl markedly reduced cyclic AMP (I)-induced morphol. differentiation of NB cells, but not of glioma cells. Acute treatment of

NB cells (1 μM) and glioma cells (0.3 μM) with MeHgCl increased the intracellular level of I. Chronic treatment of glioma cells with MeHgCl reduced the response of PGE1 [745-65-3]-sensitive adrenylate cyclase [9012-42-4], but chronic treatment of NB cells did not produce such an effect. The response of dopamine- and norepinephrine-sensitive adenylate cyclases in NB cells did not change after acute or chronic treatment with MeHgCl. Chronic and acute treatment of glioma cells with low concns. (0.05-0.1 μM) of MeHgCl produced marked changes in the amts. and net I-dependent and -independent phosphorylation profiles of specific proteins. Chronic treatment of NB cells (0.1 and 0.2 μM) did not produce any significant alterations in the amts. of specific proteins, but it caused marked changes in the I-dependent and -independent phosphorylation levels of cellular proteins. The morphol. and doubling time of chronically treated glioma and NB cells are similar to those of untreated cells. Thus, cultures of NB and glioma cells could be used as sensitive biol. assay for investigating the effects of those environmental pollutants which are known to cause or which have potential to cause neurol. disorders.

SO NATO Conference Series I: Ecology (1983), 5A(In Vitro Toxic. Test. Environ. Agents: Curr. Future Possibilities, Pt. A), 421-72
CODEN: NCSEDQ; ISSN: 0197-4475

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IT 56-40-6, biological studies 56-86-0, biological studies 60-92-4
7782-50-5, biological studies 9012-42-4
RL: BIOL (Biological study)
(of glioma and neuroblastoma cells, methylmercury effect on)

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